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NEWS	1		Web Page for STN Seminar Schedule - N. America
NEWS	2	JAN 08	CHEMLIST enhanced with New Zealand Inventory of Chemicals
NEWS	3	JAN 16	CA/CAplus Company Name Thesaurus enhanced and reloaded
NEWS	4	JAN 16	IPC version 2007.01 thesaurus available on STN
NEWS	5	JAN 16	WPIDS/WPINDEX/WPIX enhanced with IPC 8 reclassification data
NEWS	6	JAN 22	CA/CAplus updated with revised CAS roles
NEWS	7	JAN 22	CA/CAplus enhanced with patent applications from India
NEWS	8	JAN 29	PHAR reloaded with new search and display fields
NEWS	9	JAN 29	CAS Registry Number crossover limit increased to 300,000 in multiple databases
NEWS	10	FEB 15	PATDPASPC enhanced with Drug Approval numbers
NEWS	11	FEB 15	RUSSIAPAT enhanced with pre-1994 records
NEWS	12	FEB 23	KOREAPAT enhanced with IPC 8 features and functionality
NEWS	13	FEB 26	MEDLINE reloaded with enhancements
NEWS	14	FEB 26	EMBASE enhanced with Clinical Trial Number field
NEWS	15	FEB 26	TOXCENTER enhanced with reloaded MEDLINE
NEWS	16	FEB 26	IFICDB/IFIPAT/IFIUDB reloaded with enhancements
NEWS	17	FEB 26	CAS Registry Number crossover limit increased from 10,000 to 300,000 in multiple databases
NEWS	18	MAR 15	WPIDS/WPIX enhanced with new FRAGHITSTR display format
NEWS	19	MAR 16	CASREACT coverage extended
NEWS	20	MAR 20	MARPAT now updated daily
NEWS	21	MAR 22	LWPI reloaded
NEWS	22	MAR 30	RDISCLOSURE reloaded with enhancements
NEWS	23	APR 02	JICST-EPLUS removed from database clusters and STN
NEWS	24	APR 30	GENBANK reloaded and enhanced with Genome Project ID field
NEWS	25	APR 30	CHEMCATS enhanced with 1.2 million new records
NEWS	26	APR 30	CA/CAplus enhanced with 1870-1889 U.S. patent records
NEWS	27	APR 30	INPADOC replaced by INPADOCDB on STN
NEWS	28	MAY 01	New CAS web site launched
NEWS	29	MAY 08	CA/CAplus Indian patent publication number format defined
NEWS	30	MAY 14	RDISCLOSURE on STN Easy enhanced with new search and display fields
NEWS	31	MAY 21	BIOSIS reloaded and enhanced with archival data
NEWS	32	MAY 21	TOXCENTER enhanced with BIOSIS reload
NEWS	33	MAY 21	CA/CAplus enhanced with additional kind codes for German patents
NEWS	34	MAY 22	CA/CAplus enhanced with IPC reclassification in Japanese patents

NEWS EXPRESS	NOVEMBER 10 CURRENT WINDOWS VERSION IS V8.01c, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 25 SEPTEMBER 2006.
NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS LOGIN	Welcome Banner and News Items
NEWS IPC8	For general information regarding STN implementation of IPC 8

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FILE 'HOME' ENTERED AT 11:44:31 ON 11 JUN 2007

=> file medline, biosis

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 11:45:18 ON 11 JUN 2007

FILE 'BIOSIS' ENTERED AT 11:45:18 ON 11 JUN 2007
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=> filamin 1
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For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s filamin 1
L1 55 FILAMIN 1

=> s l1 and (binding)
L2 23 L1 AND (BINDING)

=> s l2 and (protein)
L3 18 L2 AND (PROTEIN)

=> s l3 and (encoding DNA)
L4 0 L3 AND (ENCODING DNA)

=> d l3 ti abs ibib 1-10

L3 ANSWER 1 OF 18 MEDLINE on STN
TI Dynamic interactions of Fc gamma receptor IIB with filamin-bound SHIP1 amplify filamentous actin-dependent negative regulation of Fc epsilon receptor I signaling.
AB The engagement of high affinity receptors for IgE (FcepsilonRI) generates both positive and negative signals whose integration determines the intensity of mast cell responses. FcepsilonRI-positive signals are also negatively regulated by low affinity receptors for IgG (FcgammaRIIB). Although the constitutive negative regulation of FcepsilonRI signaling was shown to depend on the submembranous F-actin skeleton, the role of this compartment in FcgammaRIIB-dependent inhibition is unknown. We show in this study that the F-actin skeleton is essential for FcgammaRIIB-dependent negative regulation. It contains SHIP1, the phosphatase responsible for inhibition, which is constitutively associated with the actin-binding protein, filamin-1.
After coaggregation, FcgammaRIIB and FcepsilonRI rapidly interact with the F-actin skeleton and engage SHIP1 and filamin-1.
Later, filamin-1 and F-actin dissociate from FcR
complexes, whereas SHIP1 remains associated with FcgammaRIIB. Based on these results, we propose a dynamic model in which the submembranous F-actin skeleton forms an inhibitory compartment where filamin-

1 functions as a donor of SHIP1 for FcgammaRIIB, which concentrate this phosphatase in the vicinity of FcepsilonRI and thereby extinguish activation signals.

ACCESSION NUMBER: 2005099407 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15661894
TITLE: Dynamic interactions of Fc gamma receptor IIB with filamin-bound SHIP1 amplify filamentous actin-dependent negative regulation of Fc epsilon receptor I signaling.
AUTHOR: Lesourne Renaud; Fridman Wolf H; Daeron Marc
CORPORATE SOURCE: Laboratoire d'Immunologie Cellulaire et Clinique, Institut National de la Sante et de la Recherche Medicale, Unite 255, Institut Biomedical des Cordeliers, Paris, France.
SOURCE: Journal of immunology (Baltimore, Md. : 1950), (2005 Feb 1) Vol. 174, No. 3, pp. 1365-73.
Journal code: 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200503
ENTRY DATE: Entered STN: 1 Mar 2005
Last Updated on STN: 16 Mar 2005
Entered Medline: 15 Mar 2005

L3 ANSWER 2 OF 18 MEDLINE on STN
TI Recovery from DNA damage-induced G2 arrest requires actin-binding protein filamin-A/actin-binding protein 280.
AB Filamin-A (filamin-1) is an actin-binding protein involved in the organization of actin networks. Our previous study shows that filamin-A interacts with BRCA2, and lack of filamin-A expression results in increased cellular sensitivity to several DNA damaging agents in melanoma cells (Yuan, Y., and Shen, Z. (2001) J. Biol. Chemical 276, 48318-48324), suggesting a role of filamin-A in DNA damage response. In this report, we demonstrated that deficiency of filamin-A results in an 8-h delay in the recovery from G2 arrest in response to ionizing radiation. However, filamin-A deficiency does not affect the initial activation of the G2/M checkpoint. We also found that filamin-A deficiency results in sustained activation of Chk1 and Chk2 after irradiation. This in turn causes a delay in the dephosphorylation of phospho-Cdc2, which is inhibitory to the G2/M transition. In addition, filamin-A-deficient M2 cells undergo mitotic catastrophe-related nuclear fragmentation after they are released from the G2 arrest. Together, these data suggest a functional role of filamin-A in the recovery from G2 arrest and subsequent mitotic cell death after DNA damage.
ACCESSION NUMBER: 2004065779 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14660646
TITLE: Recovery from DNA damage-induced G2 arrest requires actin-binding protein filamin-A/actin-binding protein 280.
AUTHOR: Meng Xiangbing; Yuan Yuan; Maestas Adrian; Shen Zhiyuan
CORPORATE SOURCE: Department of Molecular Genetics and Microbiology, MSC08 4660, 1 University of New Mexico School of Medicine, Albuquerque, New Mexico 87131-0001, USA.
CONTRACT NUMBER: R01 ES08353 (NIEHS)
SOURCE: The Journal of biological chemistry, (2004 Feb 13) Vol. 279, No. 7, pp. 6098-105. Electronic Publication: 2003-12-01.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 200403
ENTRY DATE: Entered STN: 10 Feb 2004
Last Updated on STN: 31 Mar 2004
Entered Medline: 30 Mar 2004

L3 ANSWER 3 OF 18 MEDLINE on STN

TI A mass spectrometry-based proteomic approach for identification of serine/threonine-phosphorylated proteins by enrichment with phospho-specific antibodies: identification of a novel protein, Frigg, as a protein kinase A substrate.

AB Although proteins phosphorylated on tyrosine residues can be enriched by immunoprecipitation with anti-phosphotyrosine antibodies, it has been difficult to identify proteins that are phosphorylated on serine/threonine residues because of lack of immunoprecipitating antibodies. In this report, we describe several antibodies that recognize phosphoserine/phosphothreonine-containing proteins by Western blotting. Importantly, these antibodies can be used to enrich for proteins phosphorylated on serine/threonine residues by immunoprecipitation, as well. Using these antibodies, we have immunoprecipitated proteins from untreated cells or those treated with calyculin A, a serine/threonine phosphatase inhibitor. Mass spectrometry-based analysis of bands from one-dimensional gels that were specifically observed in calyculin A-treated samples resulted in identification of several known serine/threonine-phosphorylated proteins including drebrin 1, alpha-actinin 4, and filamin-1. We also identified a protein, poly(A)-binding protein 2, which was previously not known to be phosphorylated, in addition to a novel protein without any obvious domains that we designate as Frigg. Frigg is widely expressed and was demonstrated to be a protein kinase A substrate in vitro. We identified several in vivo phosphorylation sites by tandem mass spectrometry using Frigg protein immunoprecipitated from cells. Our method should be applicable as a generic strategy for enrichment and identification of serine/threonine-phosphorylated substrates in signal transduction pathways.

ACCESSION NUMBER: 2002477520 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12239280
TITLE: A mass spectrometry-based proteomic approach for identification of serine/threonine-phosphorylated proteins by enrichment with phospho-specific antibodies: identification of a novel protein, Frigg, as a protein kinase A substrate.
AUTHOR: Gronborg Mads; Kristiansen Troels Zakarias; Stensballe Allan; Andersen Jens S; Ohara Osamu; Mann Matthias; Jensen Ole Norregaard; Pandey Akhilesh
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, Center for Experimental Bioinformatics, University of Southern Denmark, Campusvej 55, 5230 Odense M, Denmark.
CONTRACT NUMBER: CA 75447 (NCI)
SOURCE: Molecular & cellular proteomics : MCP, (2002 Jul) Vol. 1, No. 7, pp. 517-27.
Journal code: 101125647. ISSN: 1535-9476.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200304
ENTRY DATE: Entered STN: 20 Sep 2002
Last Updated on STN: 6 Apr 2003
Entered Medline: 4 Apr 2003

L3 ANSWER 4 OF 18 MEDLINE on STN

TI Amelioration of the macrothrombocytopenia associated with the murine Bernard-Soulier syndrome.

AB An absent platelet glycoprotein (GP) Ib-IX receptor results in the Bernard-Soulier syndrome and is characterized by severe bleeding and the laboratory presentation of macrothrombocytopenia. Although the macrothrombocytopenic phenotype is directly linked to an absent GP Ib-IX complex, the disrupted molecular mechanisms that produce the macrothrombocytopenia are unknown. We have utilized a mouse model of the Bernard-Soulier syndrome to engineer platelets expressing an alpha-subunit of GP Ib (GP Ibalpha) in which most of the extracytoplasmic sequence has been replaced by an isolated domain of the alpha-subunit of the human interleukin-4 receptor (IL-4Ralpha). The IL-4Ralpha/GP Ibalpha fusion is membrane expressed in Chinese hamster ovary (CHO) cells, and its expression is facilitated by the presence of human GP IX and the beta-subunit of GP Ib. Transgenic animals expressing a chimeric receptor were generated and bred into the murine Bernard-Soulier syndrome-producing animals devoid of mouse GP Ibalpha but expressing the IL-4Ralpha/GP Ibalpha fusion sequence. The characterization of these mice revealed a 2-fold increase in circulating platelet count and a 50% reduction in platelet size when compared with platelets from the mouse model of the Bernard-Soulier syndrome. Immunoprecipitation confirmed that the IL-4Ralpha/GP Ibalpha subunit interacts with filamin-1 and 14-3-3zeta, known binding proteins to the GP Ibalpha cytoplasmic tail. Mice expressing the chimeric receptor retain a severe bleeding phenotype, confirming a critical role for the GP Ibalpha extracytoplasmic domain in hemostasis. These results provide in vivo insights into the structural elements of the GP Ibalpha subunit that contribute to normal megakaryocyte maturation and thrombopoiesis.

ACCESSION NUMBER: 2002442472 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12200373
TITLE: Amelioration of the macrothrombocytopenia associated with the murine Bernard-Soulier syndrome.
AUTHOR: Kanaji Taisuke; Russell Susan; Ware Jerry
CORPORATE SOURCE: Roon Center for Arteriosclerosis and Thrombosis, Division of Experimental Hemostasis and Thrombosis, Department of Molecular and Experimental Medicine, The Scripps Research Institute, 10550 N Torrey Pines Road, La Jolla, CA 92037, USA.
CONTRACT NUMBER: HL 50545 (NHLBI)
SOURCE: Blood, (2002 Sep 15) Vol. 100, No. 6, pp. 2102-7.
Journal code: 7603509. ISSN: 0006-4971.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200210
ENTRY DATE: Entered STN: 30 Aug 2002
Last Updated on STN: 26 Oct 2002
Entered Medline: 24 Oct 2002

L3 ANSWER 5 OF 18 MEDLINE on STN

TI Interaction between platelet glycoprotein Ibalpha and filamin-1 is essential for glycoprotein Ib/IX receptor anchorage at high shear.

AB The interaction of the glycoprotein (GP) Ib-V-IX receptor complex with the membrane skeleton of platelets is dependent on a specific interaction between the cytoplasmic tail of GPIbalpha and filamin-1 . This interaction has been proposed to regulate key aspects of platelet function, including the ligand binding of GPIb-V-IX and the ability of the cells to sustain adhesion to von Willebrand factor (vWf) under high shear. In this study we have examined sequences in the

GPIbalpha intracellular domain necessary for interaction of the receptor with filamin-1. We have identified two adjacent sequences involving amino acids 557-568 and 569-579 of the GPIbalpha cytoplasmic domain that are critical for normal association between the receptor complex and filamin-1. Under flow conditions, Chinese hamster ovary (CHO) cells expressing these two mutant receptors exhibited an increase in translocation velocity that was associated with increased cell detachment from the vWf matrix at high shear. The shear-dependent acceleration in velocity of mutant Delta557-568 and Delta569-579 CHO cells was associated with a critical defect in receptor anchorage, evident from significant extraction of GPIb-IX from the CHO cell membrane at high shear. These studies define a critical role for amino acids within the 557-579 sequence of GPIbalpha for interaction with filamin-1.

ACCESSION NUMBER: 2002079661 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11700320
TITLE: Interaction between platelet glycoprotein Ibalpha and filamin-1 is essential for glycoprotein Ib/IX receptor anchorage at high shear.
AUTHOR: Williamson David; Pikovski Inna; Cranmer Susan L; Mangin Pierre; Mistry Nayna; Domagala Teresa; Chehab Sam; Lanza Francois; Salem Hatem H; Jackson Shaun P
CORPORATE SOURCE: Australian Centre for Blood Diseases, Department of Medicine, Monash University, Box Hill Hospital, Victoria 3128, Australia.
SOURCE: The Journal of biological chemistry, (2002 Jan 18) Vol. 277, No. 3, pp. 2151-9. Electronic Publication: 2001-11-07.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200202
ENTRY DATE: Entered STN: 28 Jan 2002
Last Updated on STN: 5 Jan 2003
Entered Medline: 13 Feb 2002

L3 ANSWER 6 OF 18 MEDLINE on STN
TI Interaction with BRCA2 suggests a role for filamin-1 (hsFLNa) in DNA damage response.
AB The BRCA2 tumor suppressor plays significant roles in DNA damage response. The human actin binding protein filamin-1 (hsFLNa, also known as ABP-280) participates in orthogonal actin network, cellular stress responses, signal transduction, and cell migration. Through a yeast two-hybrid system, an in vitro binding assay, and in vivo co-immunoprecipitations, we identified an interaction between BRCA2 and hsFLNa. The hsFLNa binding domain of BRCA2 was mapped to an internal conserved region, and the BRCA2-interacting domain of hsFLNa was mapped to its C terminus. Although hsFLNa is known for its cytoplasmic functions in cell migration and signal transduction, some hsFLNa resides in the nucleus, raising the possibility that it participates in DNA damage response through a nuclear interaction with BRCA2. Lack of hsFLNa renders a human melanoma cell line (M2) more sensitive to several genotoxic agents including gamma irradiation, bleomycin, and ultraviolet-c light. These results suggest that BRCA2/hsFLNa interaction may serve to connect cytoskeletal signal transduction to DNA damage response pathways.

ACCESSION NUMBER: 2001698266 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11602572
TITLE: Interaction with BRCA2 suggests a role for filamin-1 (hsFLNa) in DNA damage response.
AUTHOR: Yuan Y; Shen Z

CORPORATE SOURCE: Department of Molecular Genetics and Microbiology, University of New Mexico School of Medicine, Albuquerque, New Mexico 87131, USA.
CONTRACT NUMBER: ES08353 (NIEHS)
SOURCE: The Journal of biological chemistry, (2001 Dec 21) Vol. 276, No. 51, pp. 48318-24. Electronic Publication: 2001-10-15.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200201
ENTRY DATE: Entered STN: 18 Dec 2001
Last Updated on STN: 5 Jan 2003
Entered Medline: 31 Jan 2002

L3 ANSWER 7 OF 18 MEDLINE on STN
TI The small GTPase RalA targets filamin to induce filopodia.
AB The Ras-related small GTPases Rac, Rho, Cdc42, and RalA bind filamin, an actin filament-crosslinking protein that also links membrane and other intracellular proteins to actin. Of these GTPases only RalA binds filamin in a GTP-specific manner, and GTP-RalA elicits actin-rich filopods on surfaces of Swiss 3T3 cells and recruits filamin into the filopodial cytoskeleton. Either a dominant negative RalA construct or the RalA-binding domain of filamin 1 specifically block Cdc42-induced filopod formation, but a Cdc42 inhibitor does not impair RalA's effects, which, unlike Cdc42, are Rac independent. RalA does not generate filopodia in filamin-deficient human melanoma cells, whereas transfection of filamin 1 restores the functional response. RalA therefore is a downstream intermediate in Cdc42-mediated filopod production and uses filamin in this pathway.
ACCESSION NUMBER: 1999162568 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10051605
TITLE: The small GTPase RalA targets filamin to induce filopodia.
AUTHOR: Ohta Y; Suzuki N; Nakamura S; Hartwig J H; Stossel T P
CORPORATE SOURCE: Division of Biochemistry and Cellular Biology, National Institute of Neuroscience, National Center of Neurology and Psychiatry, 4-1-1 Ogawahigashi, Kodaira, Tokyo 187, Japan.. ohta@ncnaxp.ncnp.go.jp
CONTRACT NUMBER: HL54145 (NHLBI)
HL56252 (NHLBI)
HL56949 (NHLBI)
+
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1999 Mar 2) Vol. 96, No. 5, pp. 2122-8.
Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199904
ENTRY DATE: Entered STN: 26 Apr 1999
Last Updated on STN: 3 Mar 2000
Entered Medline: 15 Apr 1999

L3 ANSWER 8 OF 18 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI Dynamic interactions of Fcgamma receptor IIB with filamin-bound SHIP1 amplify filamentous actin-dependent negative regulation of Fcepsilon

receptor I signaling.

AB The engagement of high affinity receptors for IgE (FeepsilonRI) generates both positive and negative signals whose integration determines the intensity of mast cell responses. FcepsilonRI-positive signals are also negatively regulated by low affinity- receptors for IgG (FcgammaRIIB). Although the constitutive negative regulation of FcepsilonRI signaling was shown to depend on the submembranous F-actin skeleton, the role of this compartment in FcgammaRIIB -dependent inhibition is unknown. We show in this study that the F-actin skeleton is essential for FcgammaRIIB -dependent negative regulation. It contains SHIP1,. the phosphatase responsible for inhibition. which is constitutively associated with the actin-binding protein. filamin-1.
After coaggregation.. FegammaRIIB and FcepsilonRI rapidly interact with the F-actin skeleton and engage SHIP1 and filamin-1.
Later. filamin-1 and F-actin dissociate from Fell complext, whereas SHIP1 remains associated %vith FcgammaRIIB. Based on these results. we propose a dynamic model in which the submembranous F-actin skeleton forms an inhibitory compartment where filamin-1 functions as a donor of SHIP1 for FcgammaRIIB, which concentrate this phosphatase in the vicinity of FcepsilonR1 and thereby extinguish activation signals.

ACCESSION NUMBER: 2005:162785 BIOSIS
DOCUMENT NUMBER: PREV200500162456
TITLE: Dynamic interactions of Fcgamma receptor IIB with filamin-bound SHIP1 amplify filamentous actin-dependent negative regulation of Fcepsilon receptor I signaling.
AUTHOR(S): Lesourne, Renaud; Fridman, Wolf H.; Daeron, Marc [Reprint Author]
CORPORATE SOURCE: Unite Allergol Mol and CellulaireDept Immunol, Inst Pasteur, 25 Rue Dr Roux, F-75015, Paris, France daeron@pasteur.fr
SOURCE: Journal of Immunology, (February 1 2005) Vol. 174, No. 3, pp. 1365-1373. print.
ISSN: 0022-1767 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 27 Apr 2005
Last Updated on STN: 27 Apr 2005

L3 ANSWER 9 OF 18 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

TI Recovery from DNA damage-induced G2 arrest requires actin-binding protein filamin-A/actin-binding protein 280.

AB Filamin-A (filamin-1) is an actin-binding protein involved in the organization of actin networks. Our previous study shows that filamin-A interacts with BRCA2, and lack of filamin-A expression results in increased cellular sensitivity to several DNA damaging agents in melanoma cells (Yuan, Y., and Shen, Z. (2001) J. Biol. Chemical 276, 48318-48324), suggesting a role of filamin-A in DNA damage response. In this report, we demonstrated that deficiency of filamin-A results in an 8-h delay in the recovery from G2 arrest in response to ionizing radiation. However, filamin-A deficiency does not affect the initial activation of the G2/M checkpoint. We also found that filamin-A deficiency results in sustained activation of Chk1 and Chk2 after irradiation. This in turn causes a delay in the dephosphorylation of phospho-Cdc2, which is inhibitory to the G2/M transition. In addition, filamin-A-deficient M2 cells undergo mitotic catastrophe-related nuclear fragmentation after they are released from the G2 arrest. Together, these data suggest a functional role of filamin-A in the recovery from G2 arrest and subsequent mitotic cell death after DNA damage.

ACCESSION NUMBER: 2004:182199 BIOSIS
DOCUMENT NUMBER: PREV200400186117
TITLE: Recovery from DNA damage-induced G2 arrest requires actin-binding protein filamin-A/actin-binding protein 280.

AUTHOR(S): Meng, Xiangbing; Yuan, Yuan; Maestas, Adrian; Shen, Zhiyuan [Reprint Author]
CORPORATE SOURCE: Department of Molecular Genetics and Microbiology, University of New Mexico School of Medicine, MSC08 4660, 1, Albuquerque, NM, 87131-0001, USA
zshen@salud.unm.edu
SOURCE: Journal of Biological Chemistry, (February 13 2004) Vol. 279, No. 7, pp. 6098-6105. print.
CODEN: JBCHA3. ISSN: 0021-9258.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 7 Apr 2004
Last Updated on STN: 7 Apr 2004

L3 ANSWER 10 OF 18 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
TI A mass spectrometry-based proteomic approach for identification of serine/threonine-phosphorylated proteins by enrichment with phospho-specific antibodies: Identification of a novel protein, Frigg, as a protein kinase a substrate.
AB Although proteins phosphorylated on tyrosine residues can be enriched by immunoprecipitation with anti-phosphotyrosine antibodies, it has been difficult to identify proteins that are phosphorylated on serine/threonine residues because of lack of immunoprecipitating antibodies. In this report, we describe several antibodies that recognize phosphoserine/phosphothreonine-containing proteins by Western blotting. Importantly, these antibodies can be used to enrich for proteins phosphorylated on serine/threonine residues by immunoprecipitation, as well. Using these antibodies, we have immunoprecipitated proteins from untreated cells or those treated with calyculin A, a serine/threonine phosphatase inhibitor. Mass spectrometry-based analysis of bands from one-dimensional gels that were specifically observed in calyculin A-treated samples resulted in identification of several known serine/threonine-phosphorylated proteins including drebrin 1, alpha-actinin 4, and filamin-1. We also identified a protein, poly(A)-binding protein 2, which was previously not known to be phosphorylated, in addition to a novel protein without any obvious domains that we designate as Frigg. Frigg is widely expressed and was demonstrated to be a protein kinase A substrate in vitro. We identified several in vivo phosphorylation sites by tandem mass spectrometry using Frigg protein immunoprecipitated from cells. Our method should be applicable as a generic strategy for enrichment and identification of serine/threonine-phosphorylated substrates in signal transduction pathways.
ACCESSION NUMBER: 2002:563869 BIOSIS
DOCUMENT NUMBER: PREV200200563869
TITLE: A mass spectrometry-based proteomic approach for identification of serine/threonine-phosphorylated proteins by enrichment with phospho-specific antibodies: Identification of a novel protein, Frigg, as a protein kinase a substrate.
AUTHOR(S): Gronborg, Mads; Kristiansen, Troels Zakarias; Stensballe, Allan; Andersen, Jens S.; Ohara, Osamu; Mann, Matthias [Reprint author]; Jensen, Ole Norregaard [Reprint author]; Pandey, Akhilesh
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, Center for Experimental Bioinformatics, University of Southern Denmark, Campusvej 55, 5230, Odense M, Denmark
mann@bmb.sdu.dk; jenseno@bmb.sdu.dk; pandey@cebi.sdu.dk
SOURCE: Molecular and Cellular Proteomics, (July, 2002) Vol. 1, No. 7, pp. 517-527. print.
ISSN: 1535-9476.
DOCUMENT TYPE: Article

LANGUAGE:	English
OTHER SOURCE:	Genbank-BAA31643; EMBL-BAA31643; DDBJ-BAA31643; Genbank-NP001447; EMBL-NP001447; DDBJ-NP001447; Genbank-NP001605; EMBL-NP001605; DDBJ-NP001605; Genbank-NP002464; EMBL-NP002464; DDBJ-NP002464; Genbank-NP004386; EMBL-NP004386; DDBJ-NP004386; Genbank-NP004915; EMBL-NP004915; DDBJ-NP004915; Genbank-Q15097; EMBL-Q15097; DDBJ-Q15097
ENTRY DATE:	Entered STN: 30 Oct 2002 Last Updated on STN: 30 Oct 2002